

A Brief Report on Thyroid in Physiology **Varsha Datrika***

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A Brief Report

Thyroid hormone regulation begins in the hypothalamus. The hypothalamus sends thyrotropin-releasing hormone (TRH) to the anterior pituitary gland through the hypothalamic-hypophyseal portal system. TRH activates the anterior pituitary's thyrotropin cells to release thyroid-stimulating hormone (TSH). TRH is a peptide hormone produced by the cell bodies of the hypothalamic periventricular nucleus (PVN). The neurosecretory neurons in these cell bodies project down to the hypophyseal portal circulation, where TRH can accumulate before reaching the anterior pituitary.

TRH is a tropic hormone, which means it impacts cells indirectly via stimulating other endocrine glands. It interacts to TRH receptors on the anterior pituitary gland, triggering a G-protein coupled receptor-mediated signal cascade. When Gq protein is activated, phosphoinositide-specific phospholipase C is activated as well (PLC). PLC converts phosphatidylinositol 4,5-P(PIP) to inositol 1,4,5-triphosphate (IP) and 1,2-diacylglycerol through hydrolysis (DAG). These second messengers mobilize intracellular calcium reserves and activate protein kinase C, which results in TSH transcription and downstream gene activation. Through the hypothalamic-pituitary-prolactin axis, TRH has a non-tropic influence on the pituitary gland. TRH activates lactotropic cells in the anterior pituitary to generate prolactin directly as a non-tropic hormone. Other chemicals that increase prolactin secretion include serotonin, gonadotropin-releasing hormone, and oestrogen. Breast tissue growth and lactation are both caused by prolactin. TSH is released into the bloodstream and binds to the thyroid-releasing hormone receptor (TSH-R) on the thyroid follicular cell's basolateral side. The TSH-R is a Gs-protein coupled receptor that causes adenyl cyclase to activate and intracellular levels of cAMP to rise. Protein kinase A is (PKA). PKA modifies the activities when levels cAMP rise PKA.

PKA modifies the activities of several proteins by phosphorylating them. Below are the five steps of thyroid synthesis. Thyroglobulin Synthesis: Thyrocytes in thyroid follicles generate a protein known as thyroglobulin (TG). TG is a precursor protein that is kept in the lumen of follicles and does not include any iodine. The rough endoplasmic reticulum produces it. It is packed into vesicles by the Golgi apparatus and subsequently exocytose into the follicular lumen.

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Iodide absorption: Kinase of protein Increased activity of basolateral Na⁺ symporters, which are driven by Na⁺-K⁺-ATPase, brings iodide from the circulation into the thyrocytes, is caused by phosphorylation. Iodide then diffuses from the cell's basolateral side to the apex, where the pendrin transporter transports it into the colloid.

Thyroglobulin iodination: Protein kinase A also phosphorylates and activates thyroid peroxidase (TPO). Oxidation, organification, and coupling reaction are the three functions of TPO.

TPO converts iodide (I⁻) to iodine by oxidizing it with hydrogen peroxide. TPO is produced by the apical enzyme NADPH-oxidase, which produces hydrogen peroxide.

TPO connects the tyrosine residues of the thyroglobulin protein. Monoiodotyrosine (MIT) and diiodotyrosine (DIT) are produced (DIT). MIT has a single iodine-containing tyrosine residue, while DIT has two iodine-containing tyrosine residues.

TPO couples iodinated tyrosine residues to produce triiodothyronine (T₃) and tetra iodothyronine (T₄) (T₄). T₃ is formed when MIT and DIT combine, while T₄ is formed when two DIT molecules combine. Thyroid hormones are linked to thyroglobulin and stored in the follicular lumen as thyroglobulin.